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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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Jiangchun Xu

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EXAMINER

UNGAR, SUSAN NMN

ART UNIT

PAPER NUMBER

1642

DATE MAILED: 06/16/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

# Office Action Summary

Application No.

09/780,669

Applicant(s)

XU ET AL.

Examiner

Susan Ungar

Art Unit

1642

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

## Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

## Status

- 1) ☐ Responsive to communication(s) filed on 08 May 2006.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

## Disposition of Claims

- 4) ☐ Claim(s) 18, 21 and 22 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☐ Claim(s) 18, 21 and 22 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

## Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

## Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.

## Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)  
Paper No(s)/Mail Date \_\_\_\_\_
- 4) ☐ Interview Summary (PTO-413)  
Paper No(s)/Mail Date. \_\_\_\_\_
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: \_\_\_\_\_

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submissions filed on May 8, 2006, August 8, 2005 are acknowledged and have been entered. Claim 18 was amended and claims 19-20 have been canceled.
2. Claims 18 and 21-22 are pending and currently under examination.
3. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

***New Grounds of Rejection***

***Claim Rejections - 35 USC § 112***

4. The following is a quotation of the first paragraph of 35 U.S.C. 112:  
  
The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.
5. Claims 18, 21-22 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention.

The claims are drawn to a composition comprising monoclonal antibodies/monoclonal antibodies and antigen-binding fragments thereof that

specifically bind to amino acid residues 120-139 151-169, 165-184 of SEQ ID NO:114, wherein the composition is effective for inhibiting tumor growth.

The specification provides general teachings that prostate specific polypeptides isolated from a prostate tumor cDNA library are useful for diagnosing prostate cancer, useful as targets for treatment of prostate cancer (p. 1, lines 25-27) and teaches that the present invention provides pharmaceutical compositions that comprise an antibody or antigen-binding fragment that specifically binds to a polypeptide of the present invention (p. 5, lines 26-29). It is noted that inherent in a pharmaceutical composition is the *in vivo* use thereof for the treatment of disease. The specification further provides methods for inhibiting the development of a cancer in a patient comprising administering a pharmaceutical composition as set forth in the specification (p. 6, lines 24-28). Further, the specification teaches methods for determining the presence or absence of cancer, preferably prostate cancer comprising contacting a biological sample obtained from a patient with a binding agent that binds to a polypeptide of the invention, detecting the amount of polypeptide bound and comparing said amount with a predetermined cut-off value (para bridging pages 7-8), provides methods of monitoring cancer progression with said binding agent (p. 8). It is noted that none of the general teachings are drawn in particular to SEQ ID NO: 114.

As drawn specifically to SEQ ID NO:114, the specification exemplifies methods of isolating cDNA encoding prostate-specific polypeptides from a prostate tumor cDNA library (p. 120) wherein NI-1862, which is the polynucleotide (SEQ ID NO:111) which encodes the polypeptide SEQ ID NO:114 (p. 14, lines 20-30 and p. 124, lines 8-15), was isolated. The tissue specificity of the mRNA encoding SEQ ID NO:114 was determined (p. 127, lines 1-8), wherein

it was found that the mRNA encoding SEQ ID NO:114 was found to be over-expressed in 60% of the prostate tumors assayed/overexpressed in three prostate tumors as well as expressed in normal prostate, colon and kidney (p. 127, lines 20-22), wherein the mRNA encoding SEQ ID NO:114 was found to be expressed at high levels in prostate tumor and normal prostate and in low levels in normal large intestine (p. 129, lines 4-6).

One cannot extrapolate the teaching of the specification to the enablement of the claims because the claims are drawn to a composition comprising monoclonal antibodies to SEQ ID NO:114 wherein no function has been established for SEQ ID NO:114 and wherein it appears that the only function contemplated in the specification for the claimed antibodies to SEQ ID NO:114 is their use in the diagnosis and treatment of cancer/prostate cancer. For the reasons set forth below, neither of these functions, using monoclonal antibodies to SEQ ID NO:114 is enabled by the specification or the claims as originally filed wherein it cannot be determined from the specification whether the target antigen, that is the polypeptide encoded by SEQ ID NO:111, (1) is in fact even produced by any cancer cell (2) is differentially expressed in the cancer cell compared to control so that it can be used for diagnosis, (3) is presented by the cell in such a way as to permit the antibody to bind to the target *in vivo*, (4) useful for treatment.

(1) As drawn to whether or not the protein encoded by SEQ ID NO:111 is actually produced by any cancer cell and therefore is available for targeting, it is noted that no data drawn to the function or expression of the protein encoded by this novel polynucleotide sequence has been provided, thus one cannot extrapolate the teaching of the specification to the enablement of the claims because it is well known in the art that the regulation of mRNA translation is one of the major

regulatory steps in the control of gene expression, Jansen, et al, 1995, *Pediatric Res.*, 37(6):681-686). Further, those of skill in the art, recognize that expression of mRNA, specific for a tissue type, does not dictate nor predict the translation of such mRNA into a polypeptide. For example, Alberts et al. (*Molecular Biology of the Cell*, 3rd edition, 1994, page 465) teach that translation of ferritin mRNA into ferritin polypeptide is blocked during periods of iron starvation. Likewise, if excess iron is available, the transferrin receptor mRNA is degraded and no transferrin receptor polypeptide is translated. Many other proteins are regulated at the translational level rather than the transcriptional level. For instance, Shantz and Pegg (*Int J of Biochem and Cell Biol.*, 1999, Vol. 31, pp. 107-122) teach that ornithine decarboxylase is highly regulated in the cell at the level of translation and that translation of ornithine decarboxylase mRNA is dependent on the secondary structure of the mRNA and the availability of eIF-4E, which mediates translation initiation. McClean and Hill (*Eur J of Cancer*, 1993, vol. 29A, pp. 2243-2248) teach that p-glycoprotein can be overexpressed in CHO cells following exposure to radiation, without any concomitant overexpression of the p-glycoprotein mRNA. In addition, Fu et al (*EMBO Journal*, 1996, Vol. 15, pp. 4392-4401) teach that levels of p53 protein expression do not correlate with levels of p53 mRNA levels in blast cells taken from patients with acute myelogenous leukemia, said patients being without mutations in the p53 gene. As drawn specifically to cancer, Brennan et al (*Journal of Autoimmunity*, 1989, vol. 2 suppl., pp. 177-186) teach that high levels of the mRNA for TNF alpha were produced in synovial cells, but that levels of the TNF alpha protein were undetectable. Thus, protein translation is not necessarily contingent on mRNA expression due to the multitude of homeostatic factors affecting transcription and translation. Therefore, it cannot be predicted

from the information in the specification that in fact protein is produced from the identified SEQ ID NO:111 in any cancer cell. In the absence of production of the protein, it is absolutely clear that one would not know how to use the claimed invention for diagnosis or treatment of any cancer cell.

(2) If it were to be found that the encoded protein is in fact produced, as drawn to whether or not the protein encoded by SEQ ID NO:111, is differentially expressed in the cancer cell compared to control so that it can be used for targeting for diagnosis, it is noted that no data drawn to the protein expression levels of this novel polynucleotide sequence has been provided for any cancer cell, thus one cannot extrapolate the teaching of the specification to the enablement of the claim because the specification clearly teaches only that the polynucleotide, SEQ ID NO:111, is an RNA which has been identified using subtraction techniques in a cDNA prostate cancer library and has been found to be highly expressed in both normal and tumor prostate tissue, as well as in normal colon, kidney and intestine tissues and that the mRNA is apparently overexpressed in 60% of prostate tumor tissue assayed.

In particular, as drawn to the nexus between RNA and protein expression for diagnosis of a cancer cell, evidence abounds in which protein levels do not correlate with steady-state mRNA levels or alterations in mRNA levels in both cancer and normal cell types. For instance, once again, Brennan et al (Journal of Autoimmunity, 1989, vol. 2 suppl., pp. 177-186) teach that high levels of the mRNA for TNF alpha were produced in synovial cells, but that levels of the TNF alpha protein were undetectable, Zimmer (Cell Motility and the Cytoskeleton, 1991, vol. 20, pp. 325-337) teaches that there is no correlation between the mRNA level of calcium-modulated protein S100 alpha and the protein level, indicating

that S100 protein is post-transcriptionally regulated. Eriksson et al (Diabetologia, 1992, vol. 35, pp. 143-147) teach that no correlation was observed between the level of mRNA transcript from the insulin-responsive glucose transporter gene and the protein encoded thereby. Hell et al (Laboratory Investigation, 1995, Vol. 73, pp. 492-496) teach that cells in all types of Hodgkin's disease exhibited high levels of bcl-2 mRNA, while the expression of the Bcl-2 protein was not homogenous to said cells. Powell et al (Pharmacogenesis, 1998, Vol. 8, pp. 411-421) teach that mRNA levels for cytochrome P450 E1 did not correlate with the level of corresponding protein, and conclude that the regulation of said protein is highly complex. Carrere et al (Gut, 1999, vol. 44, pp. 55-551) teach an absence of correlation between protein and mRNA levels for the Reg protein. Vallejo et al (Biochimie, 2000, vol. 82, pp. 1129-1133) teach that no correlation was found between NRF-2 mRNA and protein levels suggesting post-transcriptional regulation of NRF-2 protein levels. Guo et al (Journal of Pharmacology and Experimental Therapeutics, 2002, vol. 300, pp. 206-212) teach that Oatp2 mRNA levels did not show a correlation with Oatp2 protein levels, suggesting that regulation of the Oatp2 protein occurs at both the transcriptional and post-translational level. These references serve to demonstrate that levels of RNAs cannot be relied upon to anticipate levels of protein. Further, Jang et al (Clinical and Experimental Metastasis, 1997, vol. 15, pp. 469-483) teach that further studies are necessary to determine if changes in protein levels track with changes in mRNA levels for metastasis associated genes in murine tumor cells, thus providing further evidence that one of skill in the art cannot anticipate that the level of a specific mRNA expressed by a cell will be paralleled at the protein level due to complex homeostatic factors controlling translation and post-translational



modification. Thus, in the absence of objective evidence demonstrating that not only the RNA, but also the protein is differentially expressed in cancer cells, one would not be able to predictably diagnose any cancer cell by immunoassay for protein encoded by SEQ ID NO:111.

(3) If it were to be found that the encoded protein is in fact expressed and differentially expressed, as drawn to whether or not the protein encoded by SEQ ID NO:111 is presented by a cancer cell, *in vivo*, in such a way as to permit the antibody to bind to the target SEQ ID NO:114, it is noted that no data drawn to the protein localization of this novel polynucleotide sequence has been provided. Given that neither the specification nor the art of record provides information as to whether or not the particular protein encoded by SEQ ID NO:111 is an intracellular, secreted or transmembrane protein, one of ordinary skill could not predictably determine whether or not the antibody would be able to specifically target SEQ ID NO:114 on said cancer cell for any of diagnosis or treatment of the cancer cell, *in vivo*, because neither intracellular, nor secreted proteins would be presented in a way that would permit the antibody to bind to the antigen and to target the cancer cell. Further, even if the protein were presented on the cell surface, it cannot be determined from the information in the specification whether it is present in sufficient concentration on a sufficient number of cancer cells to allow for successful diagnostic or therapeutic targeting or whether the protein is shed, modulated, or down-regulated, whether the claimed antibody cross-reacts with antigens with sequence identity to the claimed protein encoded by SEQ ID NO:111. It is absolutely clear that any of shedding, expression on other cell types, cross-reactivity would substantially interfere with the contemplated and claimed uses for the claimed compositions/monoclonal antibodies and make them the

contemplated and claimed uses unpredictable. For example, how would one diagnose a cancer cell if the antibody binds to a plethora of cells, normal and cancer, rendering the cancer cell indistinguishable from the normal cell. How would one treat a cancer cell if the antibody is sequestered by epitopes shared by proteins other than the target protein on cells other than the cancer cell, how would one diagnose or treat if SEQ ID NO:114 is not present on the cell surface in sufficient concentration on a sufficient number of the cancer cells to permit effective treatment or diagnosis. In particular,

White et al. (2001, *Ann. Rev. Med.*, 2001, 52:125-145), teaches that, for a successful targeting and immunotherapy, besides specificity of the antibody for the antigen, other properties of the antigen should be considered including the following: (1) the antigen should be present on all or near all of the malignant cells to allow effective targeting and to prevent a subpopulation of antigen-negative cells from proliferating; and (2) whether the antigens are shed, modulated or internalized influences the effectiveness of the administered immunotherapy (i.e. the antibody) (p. 126, second paragraph). Additionally, antigen internalization or downregulation can cause repeat dosing to be unsuccessful due to the disappearance of the antibody target (p. 126, paragraph before last). Thus, even if SEQ ID NO:114 is expressed, differentially expressed, presented at the cell surface, it cannot be predicted if the receptor is present on a sufficient number of cancer cells, and in sufficient quantity, to allow for successful diagnostic or therapeutic targeting of cancer cells. In view of the above, one cannot predict whether SEQ ID NO:114 is expressed in sufficient amount on cancer cells such that the claimed monoclonal antibodies would function as contemplated or claimed. Additionally, it cannot be predicted whether the antigen sheds, or is

modulated, internalized, or down regulated in primary cancer cells. Thus, again, it would require undue experimentation to determine if and under what circumstances the claimed monoclonal antibodies would be useful as contemplated and claimed.

(4) If it were to be found that the encoded protein is in fact produced, differentially expressed, as drawn to whether or not monoclonal antibodies that bind to SEQ ID NO114 are useful for treatment of a cancer cell, it is noted that no data drawn to any nexus between protein expression and treatment has been provided. In particular, it is well known that the art of anti-cancer therapy is highly unpredictable, for example, Gura (Science, 1997, 278:1041-1042) teaches that researchers face the problem of sifting through potential anticancer agents to find ones promising enough to make human clinical trials worthwhile and teach that since formal screening began in 1955, many thousands of drugs have shown activity in either cell or animal models but that only 39 have actually been shown to be useful for chemotherapy (p. 1041, see first and second para). Because of the known unpredictability of the art, in the absence of experimental evidence in an appropriate animal model, with data commensurate in scope with the invention claimed, no one skilled in the art would accept the assertion that the claimed composition could be used to effectively inhibit tumor growth based only upon the expression of SEQ ID NO:111 in prostate tumor and normal prostate tissue, normal colon intestine and kidney.

Applicant is reminded that MPEP 2164.03 teaches that "the amount of guidance or direction needed to enable the invention is inversely related to the amount of knowledge in the state of the art as well as the predictability of the art. In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970). The amount of

guidance or direction refers to that information in the application, as originally filed, that teaches exactly how to make or use the invention. The more that is known in the prior art about the nature of the invention, how to make, and how to use the invention, and the more predictable the art is, the less information needs to be explicitly stated in the specification. In contrast, if little is known in the prior art about the nature of the invention and the art is unpredictable, the specification would need more detail as how to make and use the invention in order to be enabling.” Given only lack of guidance in the specification, no one skilled in the art would accept the assertion that the claimed invention would function as contemplated or as claimed based only on the information in the specification and that known in the art at the time the invention was made.

The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention will function as claimed or contemplated with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

6. If Applicant were able to overcome the rejection set forth above, claim 22 would still be rejected under 35 USC 112, first paragraph because the specification, while enabling for a composition effective for inhibiting prostate cancer tumor growth does not reasonably provide enablement for a composition effective for inhibiting tumor growth. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to practice the invention commensurate in scope with these claims.

The claims are drawn to a composition effective for inhibiting tumor growth.

This means any tumor growth, malignant or benign as well as growth of a tumor from any tissue.

The specification teaches as set forth above. To reiterate, the specification teaches that NI-1862, which is the polynucleotide (SEQ ID NO:111) which encodes the polypeptide SEQ ID NO:114 (p. 14, lines 20-30 and p. 124, lines 8-15), is found to be over-expressed in 60% of the prostate tumors assayed/overexpressed in three prostate tumors as well as expressed in normal prostate, colon and kidney (p. 127, lines 20-22), wherein the mRNA encoding SEQ ID NO:114 was found to be expressed at high levels in prostate tumor and normal prostate and in low levels in normal large intestine (p. 129, lines 4-6).

One cannot extrapolate the teaching of the specification to the scope of the claims because even if a nexus were to be determined between the amount of mRNA overexpression and protein expression, there is no teaching of any tumor, other than prostate tumor that overexpresses SEQ ID NO:111 or 114. In particular, the heterogeneity of cancers is well known in the art. Cancers comprises a broad group of malignant neoplasms divided into two categories, carcinoma and sarcoma. The carcinomas originate in epithelial tissues while sarcomas develop from connective tissues, see Tabor's Cyclopedic Medical Dictionary (1985, F.A. Davis Company, Philadelphia, p. 274). Given that not all cancers originate from the same tissue types, it is expected and known that cancers which originate from different tissue types have different structures as well as etiologies and would present differently. Thus, it would not be predictably expected that a nexus, for example drawn to a connection between SEQ ID NO:114 and prostate cancer could be extrapolated to cancer types that arose from different tissue types. Further, it is well known that even two carcinomas that present on the same organ

have significant differences in etiology and genetic constitution. For example, Busken, C et al, (Digestive Disease Week Abstracts and Itinerary Planner, 2003, abstract No:850), teach that there is a difference in COX-2 expression with respect to intensity, homogeneity, localization and prognostic significance between adenocarcinoma of the cardia and distal esophagus, suggesting that these two cancers have different etiology and genetic constitution (last five lines of the abstract). Given the above, it is clear that it is not possible to predictably extrapolate a correlation between SEQ ID NO:114 and cancer in any tumor type other than prostate cancer, based on the information in the specification and known in the art. Again, it is clear that the claimed invention is drawn to an undeveloped art. In the case of undeveloped art, the teachings of the MPEP are clear. MPEP 2164.03 teaches that “the amount of guidance or direction needed to enable the invention is inversely related to the amount of knowledge in the state of the art as well as the predictability of the art. In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970). The amount of guidance or direction refers to that information in the application, as originally filed, that teaches exactly how to make or use the invention. The more that is known in the prior art about the nature or the invention, how to make, and how to use the invention, and the more predictable the art is, the less information needs to explicitly stated in the specification. In contrast, if little is known in the prior art about the nature of the invention and the art is unpredictable, the specification would need more detail as how to make and use the invention in order to be enabling.” Clearly given the undeveloped nature of the art of a nexus between SEQ ID NO:114 and cancer, more is required than the single example of a nexus between SEQ ID NO:114 and prostate cancer.

The specification provides insufficient guidance with regard to these issues

and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention as broadly claimed will function as claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

7. All other objections and rejections set forth in the previous Office Action are hereby withdrawn.

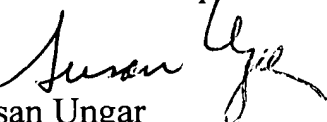
8. No claims allowed.

9. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (571) 272-0837. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Siew, can be reached at 571-272-0787. The fax phone number for this Art Unit is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Effective, February 7, 1998, the Group and/or Art Unit location of your application in the PTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Group Art Unit 1642.

  
Susan Ungar  
Primary Patent Examiner  
May 29, 2006